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Issue Date: 11 January 2007

Case No: 2006-BLA-5640
2006-BLA-5641

In the Matter of:

M.H., Widow,
Claimant

v.

Dixie Pine Coal Co.,
Employer

And

Director, Office of Workers' Compensation Programs,
Party-In-Interest

DECISION AND ORDER DENYING BENEFITS ON LIVING MINER'S AND SURVIVOR'S CLAIMS1

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. Section 901 et seq. (the Act). In accordance with the Act and the regulations issued thereunder, the case was referred by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awardable to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners who were totally disabled at the time of their deaths (for claims filed prior to January 1, 1982), or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as "black lung."

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1 Title 20 C.F.R. § 725.477(b) provides that "A decision and order shall contain . . . the names of the parties . . . ." In spite of this regulatory requirement, and the fact that by statute and regulation, black lung and longshore hearings are open to the public, the Department of Labor has decided that in order to limit a claimant's "exposure" on the Internet, it will avoid referring directly to the claimant's name in decisions and other orders that are required to be posted on the DOL web site on or after August 1, 2006. Thus, as directed by Chief Administrative Law Judge John M. Vittone, I am required to refer to the Claimant and members of her family by initials only.

A formal hearing was held before the undersigned on September 20, 2006, in Knoxville, Tennessee at which all parties were afforded full opportunity in accordance with the Rules of Practice and Procedure (29 C.F.R. Part 18) to present evidence and argument as provided in the Act and the regulations issued thereunder, set forth in Title 20, Code of Federal Regulations, Parts 410, 718, 725, and 727.[2] At the hearing, I admitted Director's Exhibits 1 through 97, Claimant's Exhibit 1, Employer's Exhibits 1-7 in the Miner's Claim, and Employer's Exhibits 1-5 in the Survivor's Claim. The Employer submitted its posthearing brief on December 27, 2006; the Director submitted its posthearing brief on November 27, 2006; the Claimant did not submit a posthearing brief.

I have based my analysis on the entire record, including the exhibits and representations of the parties, and given consideration to the applicable statutory provisions, regulations, and case law, and made the following findings of fact and conclusions of law.[3]

JURISIDICTION AND PROCEDURAL HISTORY

This case encompasses two claims. The deceased miner (Mr. H.) filed his claim for benefits on September 5, 2002 (DX 2). The District Director denied this claim on September 22, 2004, finding that Mr. H. did not suffer from pneumoconiosis and did not establish that he was permanently disabled as a result of a pulmonary impairment. (DX 46). Mr. H. filed a timely request for a hearing. (DX 47).

Mr. H. passed away on April 29, 2004 (DX 76), and his wife filed a claim for survivors' benefits on June 15, 2005. (DX 71). On March 18, 2006, the Director denied this claim, finding that Mr. H. did not suffer from pneumoconiosis. (DX 89). On March 24, 2006, Mrs. H. submitted a timely request for a hearing. (DX 90).

Both cases were referred to the Office of Administrative Law Judges, and a hearing was held on September 20, 2006.

ISSUES PRESENTED

The issues contested by the Employer and the Director are:

1. Whether Mr. H. had pneumoconiosis.
2. If so, whether Mr. H.'s pneumoconiosis arose out of his coal mine employment.
3. Whether Mr. H. was totally disabled.
4. If so, whether Mr. H.'s total disability was due to pneumoconiosis.

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2 The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vol. 65, No. 245 Wednesday, December 20, 2000. The revised Part 718 regulations became effective on January 19, 2001. Since the current claims were filed on September 5, 2002, and June 15, 2005, the new regulations are applicable.

3 Citations to the record of this proceeding will be abbreviated as follows: "Tr." refers to the Hearing Transcript of the September 20, 2006 hearing; "DX" refers to the Director's Exhibits; "CX" refers to Claimant's Exhibits; and "EX" refers to Employer's Exhibits.

5. Whether Mr. H's death was due to pneumoconiosis.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted and arguments made.

Background

Mr. H. was born on March 13, 1947 and completed the ninth grade in school. (DX 2). He married his wife, M., in 1964; he passed away on April 29, 2004. (DX 2, 71). Mr. H. had a 30- to 34-year history of cigarette smoking, ending in 2002. (DX 11, 78, 57, 59). Based on Mr. H.'s Social Security records, the District Director credited him with seven years and three months of coal mine employment. (DX 46). I have reviewed the Director's analysis and find that it is supported by the evidence of record, including Mr. H.'s Social Security earnings records, and I therefore agree with the Director's analysis, and credit Mr. H. with seven years and three months of coal mine employment.

Testimony by Mrs. H.

Mr. H's wife, Mrs. H., testified at the hearing on September 20, 2006. She testified that while Mr. H. was employed in the coal mines, he worked exclusively on the surface, and never underground. (Tr. 22). He began working in the coal mines shortly after they were married in 1964. (Tr. 23). The last coal mine operator for which he worked full time was Dixie Pine, as a dozer operator. (Tr. 43). After he left his full time position with Dixie Pine, he and his brother bought a coal truck and negotiated contracts with various coal mine operators, including Dixie Pine, wherein Mr. H. and his brother agreed to haul coal for the operators. (Tr. 23, 31, 33, 42). The last coal mine operator for which Mr. H. hauled coal was Dixie Pine. (Tr. 45). During the time that Mr. H. and his brother operated their coal truck, they did not have health insurance or workers' compensation insurance. (Tr. 39-40).

Mrs. H. testified that while Mr. H. was employed in the coal mines, he never collected compensation in the form of cash. (Tr. 30-31). When Mr. H. worked in the coal mines, he would cough up coal dust, and she would wash coal dust out of his clothes. (Tr. 45, 46).

In 1984, Mr. H. quit working because of kidney stones and breathing problems. (Tr. 44). However, he returned to work as a log truck operator in 1989. (Tr. 35). He never returned to work in the coal mines (Tr. 36), and in 1990 he quit working again and began collecting Social Security benefits. (Tr. 1990).

As to Mr. H.'s smoking history, Mrs. H. testified that he smoked a pack and a half a day, and quit smoking one month before he died. (Tr. 25). She stated that at the time of Mr. H.'s death, the couple had no dependent children living at home. (Tr. 27).

Testimony by B.H.

B.H., Mrs. H.'s daughter-in-law, also testified at the hearing. (Tr. 47). B.H. testified that Mrs. H. served as the guardian for her disabled son, D.H. (Tr. 47). She further testified that D.H. had previously been married, did not live in Mrs. H.'s home, and received his own Social Security check. (Tr. 47).

Responsible Operator Designation

Based on Mr. H.'s social security records, the District Director designated the Employer as the responsible operator. (DX 46, 89). The Director noted that the Employer was not the operator which most recently employed Mr. H.; however, Mr. H.'s Social Security records indicated that the two operators with which Mr. H. was most recently employed did not report earnings to constitute a full year of coal mine employment. (DX 46, 89). The Director further found that immediately preceeding his employment with those two operators, Mr. H. and his brother owned a truck, and together they hauled coal for several operators. (DX 46, 89). During this time, Mr. H. was self-employed. (DX 46, 89). However, the Director found that 70% of Mr. H.'s business was with the Employer. (DX 46, 89). Moreover, the Director noted that during this time, the Employer took deductions from Mr. H.'s earnings. (DX 46, 89).

The Employer argues that most of the evidence upon which the Director relied in making its designation came from Mr. H.'s deposition testimony. The Employer points to alleged discrepancies in Mr. H's testimony regarding the years during which he was self-employed and argues that these discrepancies discredit Mr. H.'s claims as they relate to his employment history. (Employer Brief at 39). The Employer urges that I disregard Mr. H.'s testimony and rely strictly on his Social Security records in designating the responsible operator. (Employer Brief at 39).

As the Director correctly points out, the federal black lung regulations provide for a very broad definition of "employment relationship." 20 C.F.R. § 725.493(a)(1). Section 725.493(b)(4) provides that a "self-employed operator, depending upon the facts of the case, may be considered an employee of any other operator, person, or business entity which substantially controls, supervises, or is financially responsible for the activities of the self-employed operator." Moreover, "any . . . operator who retained the right to direct, control, or supervise the work performed by the miner, or who benefited from such work, may be considered a potentially liable operator." § 725.493(a)(2).

In applying this law to the facts, I agree with the Director's finding regarding the designation of the responsible operator. I note that Mr. H. consistently testified that most of his work hauling and unloading coal during the years that he was self-employed was for Dixie Pine, and the Employer has pointed to nothing in Mr. H.'s testimony or his other statements to contradict this claim. Indeed, the fact that the Employer took deductions from Mr. H.'s pay during this period of self-employment supports Mr. H.'s testimony, and shows that the Employer retained a significant degree of control over Mr. H.'s work (DX 18, p. 25; DX 17, p. 55). I find that the evidence establishes that as a self-employed coal hauler, Mr. H. performed most of his work for the Employer, which benefited the most from Mr. H.'s work. (DX 17, p. 5-6, 10, 14-15). Finally, both Mr. and Mrs. H.'s testimony that the Employer called Mr. H. whenever it needed his services, as well as the testimony relating to Mr. H.'s concern that if he did not

comply with the Employer's request, he would not be given future work, supports my finding that the Employer retained a significant degree of control over Mr. H.'s operations. (DX 17, p. 16; Tr. 42). I therefore find that the Employer has been correctly designated as the responsible operator.

MINER'S CLAIM

Applicable Standard

For a miner, the conditions of entitlement include whether the individual (1) is a miner as defined in this section; (2) has met the requirements for entitlement to benefits by establishing pneumoconiosis, its causal relationship to coal mine employment, total disability, and contribution by the pneumoconiosis to the total disability; and (3) has filed a claim for benefits in accordance with this part. 20 C.F.R. §725.202(d). Mrs. H. must establish that Mr. H.'s condition satisfied all four elements of entitlement.

Medical Evidence

X-Ray Evidence

The following x-ray evidence has been admitted into the record in connection with Mr. H.'s claim:

Exhibit No.	***Date of X-Ray***	***Date of Reading***	***Physician/ Qualifications***[4]	***Interpretation***
DX 12	08/29/02	08/29/02	Ahmed BCR/B	1/0; p/s in all zones; emphysema
DX 14	8/29/02	04/27/04	Poulos BCR/B	No pneumoconiosis
DX 11	10/15/02	10/15/02	Baker B	1/0; p/p in upper and mid right lung and in mid left lung
DX 15	10/15/02	04/27/04	Poulos BCR/B	No pneumoconiosis
DX 11	10/15/02	10/28/02	Goldstein B	Read for quality
DX 57	07/18/03	07/18/03	Dahhan B	No pneumoconiosis
CX 1	08/25/03	08/26/03	Kiphart-Bays[5]	Moderate emphysematous

4 A "B-reader" is a physician, but not necessarily a radiologist, who has successfully completed an examination in interpreting x-ray studies conducted by, or on behalf of, the Appalachian Laboratory for Occupational Safety and Health (ALOSH). A designation of "BCR" means that the physician is "certified" in radiology or diagnostic roentgenology by the American Board of Radiology or the American Osteopathic Association.

5 Both Dr. Kiphart-Bays's and Dr. Pongdee's interpretations are part of the Middlesboro-ARH records and are therefore treatment records under 20 C.F.R. § 725.414(a)(4).

			BCR[6]	changes; small bilateral effusion; increased density at the medial right lung base maybe representing atelectasis or superimposed pneumonia; mildly prominent interstitium; mild pulmonary edema; mild cardiomegaly
DX 78, CX1	04/17/04	04/18/04	Kiphart-Bays BCR	Right middle lung collapse, the etiology of which is uncertain
DX 78, CX 1	04/22/04	04/22/04	Pongdee BCR/B[7]	Patchy infiltrate or subsegmental atelectasis in the lower right lung field and in the left lung base showing minimal interval improvement on the right and the finding on the left is a new finding since 4/17/04; mild cardiomegaly, insignificant change
DX 78, CX 1	04/25/04	04/25/04	Pongdee BCR/B	Interval increased infiltrate in the left lower lobe since 4/22/04; cardiomegaly and infiltrate or subsegmental atelectasis in the right lower lobe show no change

Pulmonary Function Studies

Exhibit No.	***Date/ Physician***	***Age/Height***	***FEV1***	***FVC***	***Effort***	***Qualifying***
DX 11	10/15/02 Baker	55/70[8]	2.57	4.21	Fair	No
DX 12	02/24/03 Unknown	55/71	2.55	4.00	Good	No

6 Dr. Kiphart-Bays's credentials are not of record; however, I take judicial notice that Dr. Kiphart-Bays is certified by the American Board of Radiology, as verified by the American Board of Medical Specialists. (http://www.abms.org/default.asp).

7 Dr. Pongdee's credentials are not of record; however, I take judicial notice that Dr. Pongdee is certified by the American Board of Radiology, as verified by the American Board of Medical Specialties. (http://www.abms.org/default.asp). In addition, I take judicial notice that Dr. Pongdee is a certified B-reader, as verified by the National Institute for Occupational Safety and Health. (http://www.oalj.dol.gov/PUBLIC/BLACK_LUNG/REFERENCES/REFERENCE_WORKS/BREAD3_08_05.HTM).

8 A majority of the pulmonary function studies performed on Mr. H. report his height as 71 inches. I find that this is the appropriate height to use in evaluating the pulmonary function studies.

DX 57	07/18/03 Dahhan	55/71	2.39	4.00	Good	No

Arterial Blood Gas Studies

Exhibit No.	***Date***	***Physician***	***pCO2***	***pO2***	***Qualifying***
DX 11	10/15/02	Baker	38	80	No[9]
DX 57	07/19/03	Dahhan	34.5	82.9	No
DX 58	04/29/04	Kaw	33 (?)[10]	60 (?)	Yes

Medical Reports

Dr. Glen Baker

At the request of the Department of Labor, Dr. Baker examined Mr. H. on October 15, 2002. (DX 11). Dr. Baker is a certified B-reader; although he is not certified by the Board of Radiologists, he is board-certified in internal medicine and pulmonary diseases.

Dr. Baker reported Mr. H.'s medical, social, and employment histories. He documented Mr. H.'s smoking history as one pack a day for 30 years, and as of the date of the examination, Mr. H. had not yet quit smoking. He noted 21 years of coal mine employment, the last five years of which were spent as a dozer operator. As to Mr. H.'s medical history, Dr. Baker reported a history of wheezing, chronic bronchitis, arthritis, heart disease, allergies, and high blood pressure. On the date of the examination, Mr. H. complained of cough with sputum production, wheezing, dyspnea, cough, hemoptysis, orthopnea, ankle edema, and shortness of breath for which he used an inhaler. Mr. H. told Dr. Baker that his medications included Clinoril, Lescol, Adalat, Cimetidine, and Vicodin.

Upon his physical examination of Mr. H., Dr. Baker found normal lungs and a rapid peripheral pulse, and sinus tachycardia rhythm in the heart. He performed a chest x-ray which he read as positive for pneumoconiosis with 1/0, p/p opacities in the upper and mid right lung zones and the mid left lung zone. Dr. Baker also administered a pulmonary function test, which revealed a mild obstructive defect, and an arterial blood gas study, the results of which were normal. In addition, Dr. Baker administered an electrocardiogram test which he read to show a sinus tachycardia rhythm, ST-T changes, and a possible old anterior septal infarct.

Based upon his examination, Dr. Baker diagnosed Mr. H. with pneumoconiosis, COPD with a mild obstructive defect, and chronic bronchitis. Dr. Baker attributed each of these

[9] Dr. Matthew A Vuskovich reviewed this pulmonary function test, and stated that it showed that on October 15, 2002, Mr. H. had the pulmonary capacity to continue working in the coal industry. He added that this statement was true even if Mr. H. were to be diagnosed with coal workers' pneumoconiosis. (DX 59).

10 Question marks appear next to the values reported in this arterial blood gas study; however, there is no explanation as to why the question marks appear.

diagnoses to coal dust exposure. He attributed the COPD and chronic bronchitis to both coal dust exposure and cigarette smoking. Dr. Baker further concluded that as a result of these diagnoses, Mr. H. was only mildly impaired and retained the capacity to return to his former job in the coal mines.

Dr. John C. Boll

By letter dated February 18, 2003, Dr. Boll, a physician with the Dayspring Family Health Clinic, submitted a report containing his opinion regarding Mr. H.'s medical condition. (DX 12). Dr. Boll reported that Mr. H. had been a patient at his clinic since 1993, and at the time that the letter was written, Mr. H. had been experiencing worsening symptoms. He was being treated with MDIs, nebulizers, steroids, and antibiotics. At the time, Dr. Boll was also considering putting Mr. H. on oxygen. Dr. Boll diagnosed Mr. H. with COPD in part due to coal dust exposure. However, Dr. Boll further noted that he had not performed any objective medical tests to confirm this diagnosis; instead, he relied solely on Mr. H.'s self-reported history.

By letter dated September 29, 2004, Dr. Boll submitted a supplemental report containing his opinion regarding Mr. H.'s medical condition. (DX 58). In this report, Dr. Boll stated that he had treated Mr. H. from October 2002 until Mr. H.'s death in April 2004. Dr. Boll reported that Mr. H. had significant pulmonary symptoms before his death with frequent visits to the clinic for exacerbation of COPD. He opined that Mr. H.'s occupational exposure to coal dust contributed to and hastened his death; however, he did not report any objective medical testing to support his opinion.

Dr. A. Dahhan

At the Employer's request, Dr. Dahhan examined Mr. H. on July 18, 2003; he also testified by deposition. (DX 57, DX 59). Dr. Dahhan is a certified B-reader; he is not certified by the Board of Radiologists, but he is board-certified in pulmonary medicine and internal medicine.

Dr. Dahhan reported Mr. H.'s medical, social, and employment histories. He documented Mr. H.'s smoking history of one pack a day for approximately 34 years. He noted fifteen years of coal mine employment as a dozer operator, terminating in 1984. As to Mr. H.'s medical history, Dr. Dahhan reported a history of hypertension, daily cough with sputum production, intermittent wheeze, and occasional chest pain. He complained of suffering dyspnea upon climbing one flight of stairs, and having to sleep on two pillows at night. Mr. H. denied being prescribed nitroglycerin, but told Dr. Dahhan that he was taking Proventil via nebulizer as needed, as well as some pills for his breathing.

Upon his physical examination of Mr. H., Dr. Dahhan found good air entry in the lungs without crepitation, rhonchi, or wheeze. Dr. Dahhan also found a normal heart. He performed an x-ray, a pulmonary function test, and an arterial blood gas study. He interpreted the x-ray to show no signs of pneumoconiosis; neither the arterial blood gas study nor the pulmonary function test, which showed a mild partially reversible obstructive defect, revealed any respiratory impairment secondary to exposure to coal mine dust.

In addition to performing the physical examination, on May 20, 2005 and August 31, 2006, Dr. Dahhan reviewed Mr. H.'s medical records, including Middlesboro-ARH records from the 2003 and 2004 admissions, Dr. Blake's autopsy report, and Dr. Boll's September 29, 2004 letter. After reviewing these records, Dr. Dahhan concluded that Mr. H. did not suffer from pneumoconiosis, but he suffered from congestive heart failure, emphysema, left ventricular impairment, and possible liver cancer. Dr. Dahhan attributed the emphysema to Mr. H.'s lengthy smoking history rather than coal dust exposure, and he attributed the congestive heart failure to left ventricular and bi-ventricular failure rather than cor pulmonale. Moreover, he concluded that in July 2003, Mr. H. was not totally disabled from a pulmonary standpoint, and that bi-ventricular heart failure and liver metastasis rather than coal dust exposure caused his death. (DX 68, 59; EX 1 – Miner's Claim).

Dr. Gregory J. Fino

At the Employer's request, Dr. Fino reviewed Mr. H.'s medical records and presented his opinion in letters dated August 24, 2006 and September 5, 2006.[11] (EX 2, 3 – Miner's Claim). Dr. Fino reviewed the Middlesboro-ARH records, Dr. Blake's autopsy report, Dr. Caffrey's autopsy review, and Dr. Boll's September 29, 2004 letter. Based upon his review of these documents, Dr. Fino did not believe that there was sufficient evidence to justify a diagnosis of simple coal workers pneumoconiosis. Instead, he believed that Mr. H.'s disabling respiratory impairment was related to cigarette smoking.

Dr. Fino was not able to say exactly what caused Mr. H.'s death. He noted that Mr. H. went to the hospital with acute renal failure; he also had significant congestive heart failure, with a left ventricular ejection fraction of 20%. He felt that it was very likely that Mr. H. died of left ventricular congestive heart failure, which is not caused by cor pulmonale. According to Dr. Fino, it appeared that renal failure and congestive heart failure were the two most significant diseases that could have contributed to Mr. H.'s death.

Treatment Records

Middlesboro-ARH Hospital Records

The record contains medical records from Mr. H's admission to Middlesboro-ARH Hospital in August 2003 with complaints of abdominal pain. (CX 1). These records contain several abdominal x-rays, CT scans, and other abdominal procedures. There is one chest x-ray which is summarized above.

The record also contains medical records from Mr. H.'s admission to Middlesboro-ARH Hospital in April 2004. (DX 58, 78). On April 17, 2004, Mr. H. was admitted complaining of cough, shortness of breath, and bilateral leg swelling. (DX 78). According to Dr. Maria Hortillosa's History and Physical dated April 17, Mr. H. had been sick for months. In her H&P, Dr. Hortillosa reported Mr. H.'s medical and social histories. She reported that Mr. H. had

11 Dr. Fino also submitted a letter dated March 28, 2005 (DX 59); however, on its evidence summary form dated September 18, 2006, the Employer did not indicate that it was relying on this letter.

suffered in the past from hypertension and COPD. She noted that Mr. H. had also smoked cigarettes in the past, but had not smoked at all in the year preceding his admission.

Upon her physical examination of Mr. H., Dr. Hortillosa noted that he looked chronically ill but did not seem to be in distress. He had wheezing in both lung fields and rales in the right posterior chest, a normal heart, and +2 to +3 bilateral pitting edema. Based on her evaluation of Mr. H.'s history and examination, Dr. Hortillosa listed her admitting diagnoses as acute exacerbation of COPD, pneumonia, hypertension, and possible congestive heart failure.

On April 18, Dr. Vincente Kaw administered an echocardiogram which revealed concentric left ventricular hypertrophy and severe left ventricular dysfunction with an ejection fraction of 20%. (DX 78).

On April 19, Dr. Dr. Kiphart-Bays read a CT scan of Mr. H.'s chest which revealed a partial collapse of the right *lower* lung lobe rather than the middle lobe, as she had diagnosed on April 17. (DX 78, CX 1). The CT scan also revealed findings consistent with congestive heart failure, two liver lesions, and bilateral adrenal hyperplasia. Dr. Kiphart-Bays also read a complete abdominal ultrasound which showed at least two echogenic liver masses. (DX 78, CX 1).

On April 20, at Dr. Hortillosa's request, Dr. Raju Vora examined Mr. H. Dr. Hortillosa requested the consultation because the April 19 CT scan showed the possibility of two metastatic lesions in the stomach. (DX 78). Dr. Vora reported that the reason for the admission to Middlesboro-ARH was cough, chronic obstructive pulmonary disease, and cor pulmonale. During the examination, Mr. H. appeared ill and was short of breath. His medical history included shortness of breath, pressure and tightness in the chest, coughing, gastrointestinal distention and discomfort, and diminished appetite.

Dr. Vora performed a chest examination, which revealed vesicular breath sounds that were decreased toward the bases. He found normal heart sounds and no rub or murmur; however, an examination of Mr. H.'s extremities revealed 3+ edema. In addition, Dr. Vora found a soft abdomen with an enlarged liver. Based on his examination, Dr. Vora concluded that Mr. H. suffered from possible metastatic disease in the liver and ordered a colonoscopy and CT-guided biopsy.

Also on April 18, Dr. Kaw administered an echocardiogram which revealed a left pleural effusion, concentric left ventricular hypertrophy, and severe left ventricular dysfunction with an ejection fraction of 20%.

On April 29, Mr. H. was transferred to Fort Sander's Hospital, and Dr. Kaw prepared a Transfer Discharge Summary. (DX 58). Dr. Kaw listed the reason for transfer as further diagnostic and therapeutic intervention for severe left ventricular dysfunction with ejection fraction of 20%. Other diagnoses included acute renal failure, COPD secondary to chronic tobacco use, and pneumonitis. He listed the reason for admission as acute exacerbation of coal-workers' pneumoconiosis. In his summary, Dr. Kaw stated that Mr. H. was referred to him because his blood pressure stayed low, and he had bilateral pedal edema and evidence of right-

sided congestive heart failure. He noted that earlier that morning, an arterial blood gas study was administered and Mr. H.'s PCO2 was 33, while his PO2 was 60. He also noted that the April 19 CT scan showed findings consistent with congestive heart failure, with a small pericardial effusion and a bilateral pleural effusion, the right greater than the left. In addition, he noted the partial collapse of the right lower lung lobe, which was likely the result of atelectasis. Finally, he noted that the liver lesions possibly represented a metastatic disease.

Upon his physical examination of Mr. H., Dr. Kaw found increased breath sounds at the base of the lungs with a mild degree of echophony and some wheezing. He found the chest wall to be symmetrically expanded, a normal heart rhythm with tachycardia, and a slightly globular, non-tender abdomen.

Autopsy Evidence

Dr. C. Blake

At Mrs. H.'s request, Dr. Blake performed an autopsy on Mr. H. on April 30, 2004. (DX 58). Dr. Blake found a mild degree of focal silicosis associated with scarring, but concluded that the silicosis was of too insignificant a degree to designate pulmonary disease as the cause of death. Instead, he found that the extreme hyalinized and swollen character of the cardiac myocytes, the presence of severe emphysema, the pulmonary scarring, and the pleural effusion were consistent with heart failure.

Dr. Blake's final diagnoses and findings included cardiomegaly with cardiomyopathy; severe pulmonary effusions, pulmonary edema and congestion; diffuse pan-lobular and bullous emphysema, moderate to severe, with heavy anthracosis and minimal silicosis; severe pulmonary interstitial fibrosis; heavy tracheobronchial mucus obstruction; pericardial effusion; and mild and moderate variable coronary atherosclerosis and aortic atherosclerosis. He stated that the proximate cause of Mr. H.'s death was severe bilateral emphysema, pulmonary fibrosis with sequelae of congestive heart failure (cor pulmonale). Dr. Blake indicated that the immediate cause of Mr. H.'s death was asphyxia of cardiopulmonary failure, with hypertensive cardiomyopathy, and that the mechanism of death was asphyxia with agonal bronchial mucus plugging and severe pulmonary edema. He stated that the initial gross dissection showed no characteristic features of bronchovascular scarring of coal workers' pneumoconiosis, and that microscopic samples would be processed to confirm his conclusion.

On his microscopic examination of the lung tissues, Dr. Blake noted that one section of the upper lobe of bronchi showed a severe emphysematous change of the surrounding parenchyma, representing panlobular bullous emphysema. He noted hemorrhage around the vessels, and extensive areas of interstitial fibrosis, with considerable aggregates of lymphocytes. There was focal anthracosis in these densely scarred areas. Dr. Blake described a few areas of very minimal or moderate anthracotic pigment, but no nodules to suggest pneumoconiosis. He subjected the slides to polarization microscopy, which showed a sprinkling of anthracotic cells in many of the densely scarred areas, from old carbon exposure. There was no tendency to bronchovascular nodules, but there was a background of severe emphysema and advanced interstitial fibrosis. Polarization microscopy showed a mild to moderate focal scattering of

sharply spiculated particles consistent with silicon particles in a few of the areas of the anthracosis. He stated that "The preserved areas of partially aerated alveoli in the severely scarred and emphysematous lungs include a rather heavy population of pigmented macrophages consistent with other environmental contamination of the lungs. Most likely the brownish pigmented macrophages within the alveoli, i.e., alveolar histiocytes resulted from cigarette smoking habit."

Dr. Blake concluded that the mild degree of focal silicosis associated with the scarring was of an insignificant degree to designate the pulmonary disease as the cause of Mr. H.'s death. He indicated that the extreme hyalinized and swollen character of the cardiac myocytes, as well as the presence of severe emphysema and pulmonary scarring, and the obvious pleural effusion, were consistent with agonal heart failure. According to Dr. Blake, the lung pathology was more consistent with chronic obstructive and cicatrizing (scarring) of the lungs, representing the agonal changes leading to cardiopulmonary failure. But the gross and microscopic criteria for coal workers' pneumoconiosis were not fulfilled by the study.

Dr. P. Raphael Caffrey

At the Employer's request, Dr. Caffrey reviewed Dr. Blake's autopsy report and the autopsy slides, and prepared a report dated October 18, 2005. (EX 4 – Miner's Claim). On his examination of the tissue slides, he noted subpleural scarring, evidence of centrilobular emphysema with areas of interstitial fibrosis, focal bullous formation, and a mild amount of anthracotic pigment subpleurally. There were histiocytes or macrophages containing brown pigment in focal areas. But there were no lesions of coal workers' pneumoconiosis, or anthracotic pigment with reticulin and/or collagen, with or without focal emphysema. There were no areas of complicated pneumoconiosis. Under polarized light, Dr. Caffrey saw a few birefringent particles, but no particles that appeared to be silica.

Dr. Caffrey's microscopic diagnosis was moderate centrilobular emphysema with focal bullous formation and associated focal interstitial fibrosis; moderately severe pulmonary edema and congestion; focal subpleural fibrosis with an area of plaque formation, and focal metaplasia with dysplasia of bronchial epithelium. In Dr. Caffrey's opinion, Mr. H. did not have coal workers' or any other occupational pneumoconiosis. He noted that the autopsy slides showed only a minimal amount of anthracotic pigment, which by itself is not synonymous with pneumoconiosis. Dr. Caffrey agreed with Dr. Blake's conclusion that the "gross and microscopic criteria of coal miners pneumoconiosis are not fulfilled in this study."

However, Dr. Caffrey disagreed with Dr. Blake's conclusion that the measurements of the heart revealed "clear cut and substantial cor pulmonale." He stated that Mr. H. had biventricular hypertrophy due to significant cardiomegaly, and what Dr. Blake described microscopically was definitely "consistent with an area of old myocardial infarction within the left ventricle." Dr. Caffrey felt that Mr. H. died a cardiac death with congestive heart failure. He did not have pneumoconiosis, and therefore that condition could not have caused, or in any way contributed to or hastened his death.

DISCUSSION

Existence of pneumoconiosis

Pneumoconiosis is defined, by regulation, as a "chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." 20 C.F.R. § 718.201. The regulations at 20 C.F.R. § 718.203(b) provide that, if it is determined that the miner suffered from pneumoconiosis and has engaged in coal mine employment for ten years or more, there is a rebuttable presumption that the pneumoconiosis arose out of such employment. If, however, it is established that the miner suffered from pneumoconiosis but worked less than ten years in the coal mines, then the claimant must establish causation by competent evidence. *Stark v. Director, OWCP,* 9 B.L.R. 1-36 (1986); *Hucker v. Consolidation Coal Co.,* 9 B.L.R. 1-137 (1986). The claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. *See, Director, OWCP v. Greenwich Collieries,* 114 S.Ct. 2251 (1995).

Because the current claim was filed after the enactment of the Part 718 regulations, the evidence will be evaluated under standards found in 20 C.F.R. Part 718. The existence of pneumoconiosis may be established by any one or more of the following methods: (1) chest x-rays; (2) autopsy or biopsy; (3) by operation of presumption; or (4) by a physician exercising sound medical judgment based on objective medical evidence. 20 C.F.R. § 718.202(a). I have independently assessed the evidence under each of these methods.

To establish the existence of pneumoconiosis, a chest x-ray must be classified as category 1, 2, 3, A, B, or C, according to the ILO-U/C classification system. A chest x-ray classified as category 0, including subcategories 0/1, 0/0, or 0/-, does not constitute evidence of pneumoconiosis. In the instant case, there are ten interpretations of seven x-rays. Dr. Ahmed, who is dually qualified, read the August 29, 2002 x-ray as positive for pneumoconiosis. However, Dr. Poulos, who is also dually qualified, read the same x-ray as negative. Dr. Baker, who is a B-reader but not a board-certified radiologist, read the October 15, 2002 x-ray as positive, but Dr. Poulos read it as negative. In addition, Dr. Dahhan, who is a B-reader but not a board-certified radiologist, read the July 18, 2003 x-ray as negative for the existence of pneumoconiosis. The August 2003 and April 2004 x-ray narrative interpretations included no findings of pneumoconiosis. Based on the preponderance of negative interpretations by well qualified readers, as well as the lack of any findings of pneumoconiosis in the most recent narrative x-rays, I find that the preponderance of the x-ray evidence does not establish the existence of pneumoconiosis.

A claimant may also establish the existence of pneumoconiosis by autopsy or biopsy evidence. 20 C.F.R. § 718.202. The record in this case contains two autopsy reports, but both Dr. Blake and Dr. Caffrey agreed that the findings did not meet the criteria to establish pneumoconiosis. Thus, I find that Mrs. H. has not established the existence of pneumoconiosis by a preponderance of the autopsy evidence.

Under § 718.202(a)(3), a determination of the existence of pneumoconiosis may also be made by using the presumptions set out in §§ 718.304, 305, or 306. Section 718.304 requires x-

ray, biopsy, or equivalent evidence of complicated pneumoconiosis, which is not present in this case. Section 718.305 is not applicable because it applies only to claims filed before January 1, 1982. Section 718.306 is only applicable in the case of a deceased miner. Since none of these presumptions is applicable, the existence of pneumoconiosis is not established under § 718.202(a)(3).

A claimant can also establish the existence of pneumoconiosis by well-reasoned, well-documented medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. *Fields v. Island Creek Coal Co.,* 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's history. *See, Hoffman v. B&G Construction Co.,* 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.,* 7 B.L.R. 1-295 (1984).

A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. *Fields, supra.* Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder of fact to decide. *Clark v. Karst-Robbins Coal Co.,* 12 B.L.R. 1-149 (1989)(en banc). Statutory pneumoconiosis is established by well-reasoned medical reports which support a finding that the miner's pulmonary or respiratory condition is significantly related to or substantially aggravated by coal dust exposure. *Wilburn v. Director, OWCP,* 11 B.L.R. 1-135 (1988). An equivocal opinion, however, may be given little weight. *Justice v. Island Creek Coal Co.,* 11 B.L.R. 1-91 (1988); *Snorton v. Zeigler Coal Co.,* 9 B.L.R. 1-106 (1986).

In this case, there are four medical opinions in the record. Dr. Baker, who examined Mr. H. on October 15, 2002, determined that he suffered from pneumoconiosis, COPD with a mild obstructive defect, and chronic bronchitis. Dr. Baker's diagnosis of pneumoconiosis was based on his positive interpretation of Mr. H.'s x-ray. But I have found that the preponderance of the x-ray evidence does not establish the existence of pneumoconiosis, and Dr. Baker offered no other medical basis for his conclusion that Mr. H. had pneumoconiosis.

Dr. Baker based his diagnosis of COPD on the results of the pulmonary function test, and described the etiology of the COPD as "coal dust exposure/cigarette smoking." Dr. Baker's diagnosis of COPD as a result of coal dust exposure falls within the definition of "legal pneumoconiosis." *See* 20 C.F.R. § 718.201(a)(2). However, Dr. Baker did not explain how he arrived at the conclusion that Mr. H.'s COPD was caused by coal dust exposure, or otherwise offer any support for his conclusory statement. Thus, I find that Dr. Baker's conclusion that Mr. H. had COPD due in part to coal dust exposure is not well-reasoned or supported by the objective medical evidence.

Dr. Baker also diagnosed Mr. H. with chronic bronchitis due to coal dust exposure and cigarette smoking. However, he based this diagnosis strictly on Mr. H.'s self-reported history of chronic bronchitis. There is no indication that Dr. Baker performed any objective medical testing to support this diagnosis. Thus, I find that Dr. Baker's diagnosis of chronic bronchitis due to coal dust exposure and cigarette smoking is not well-documented or supported, and I accord it no weight.

There is also a medical opinion from Dr. Boll, Mr. H.'s treating physician, in the record. However, Dr. Boll specifically stated that his diagnosis of COPD due in part to coal dust exposure was not supported by any objective medical tests. Nor were any treatment notes included in the record indicating that Dr. Boll had evaluated or treated Mr. H. for pneumoconiosis or COPD due to coal dust exposure. Thus, I find that Dr. Boll's opinion is not well-documented and I accord it no weight.

Dr. Dahhan examined Mr. H., and reviewed his medical records; he concluded that he did not have pneumoconiosis, or any respiratory impairment due to coal dust exposure. He attributed Mr. H.'s emphysema to cigarette smoking and not coal dust exposure. Dr. Dahhan's opinions are based on his examination findings, as well as his objective test results, and his review of Mr. H.'s medical records. I find that they were well-reasoned and supported, and I accord them significant weight.

Dr. Fino did not examine Mr. H., but he based his opinion upon his review of Mr. H.'s medical records, and concluded that Mr. H. suffered from a pulmonary impairment that was related to cigarette smoking and not pneumoconiosis. I find that Dr. Fino's conclusions are well-reasoned, and supported by the objective medical evidence, and I accord them significant weight.

Mr. H.'s treatment records include Dr. Kaw's Discharge and Transfer Summary, in which he noted that Mr. H. was admitted to Middlesboro-ARH for "exacerbation of coal workers' pneumoconiosis." However, the records pertaining to Mr. H.'s 2004 admission to Middlesboro-ARH include no indication from any other doctor that Mr. H. was ever diagnosed with coal workers' pneumoconiosis, or that any such diagnosis was made during Mr. H.'s treatment there. Indeed, Dr. Kaw's list of diagnoses did not include coal workers' pneumoconiosis, but it did include COPD secondary to tobacco use. I find that Dr. Kaw's mention of coal workers' pneumoconiosis, without more, is not probative evidence tending to establish the existence of the disease.

Neither of the autopsy reports includes findings of pneumoconiosis, either by gross or microscopic evaluation.

Giving the most weight to the opinions of Dr. Dahhan and Dr. Fino, which are consistent with the autopsy findings by Dr. Blake and Dr. Caffrey, and the lack of objective documentation in Mr. H.'s hospital treatment records, I find that Mrs. H. has not met her burden of showing the existence of pneumoconiosis by a preponderance of the reliable medical opinion evidence.

Finally, I have weighed all of the evidence relating to the existence of pneumoconiosis, like and unlike, and I find that Mrs. H. has not established by a preponderance of the medical evidence that Mr. H. had pneumoconiosis. Thus, she is not entitled to benefits in connection with Mr. H.'s living miner's claim.

SURVIVOR'S CLAIM

Applicable Standard

Because Mrs. H. filed her survivor's claim after January 1, 1982, 20 C.F.R. § 718.205(c) applies to this claim. The regulations provide that a survivor is entitled to benefits only where the miner died due to pneumoconiosis. § 718.205(a). Mrs. H. must establish that: (1) the decedent was a coal miner; (2) the decedent suffered from pneumoconiosis at the time of his death; (3) the decedent's pneumoconiosis arose out of his coal mine employment; and (4) the decedent's death was caused by pneumoconiosis or pneumoconiosis was a substantially contributing cause or factor leading to his death. All elements of entitlement must be established by a preponderance of the evidence. *Strike v. Director, OWCP*, 817 F.2d 395, 399 (7th Cir. 1987). The survivor of a miner who was totally disabled due to pneumoconiosis at the time of death, but died due to an unrelated cause, is not entitled to benefits. 718.205(c). If the principal cause of death is a medical condition unrelated to pneumoconiosis, the survivor is not entitled to benefits unless the evidence establishes that pneumoconiosis was a substantially contributing cause of the death. § 718.205(c)(4).

The Board has held that death will be considered to be due to pneumoconiosis where the cause of death is significantly related to or significantly aggravated by pneumoconiosis. *Foreman v. Peabody Coal Co.*, 8 B.L.R. 1-371 (1985). The United States Court of Appeals for the Sixth Circuit, in which the instant case arises, has held that pneumoconiosis is a substantially contributing cause of death if it hastens, even briefly, the miner's death. *See Brown v. Rock Creek Mining Corp.*, 996 F.2d 812 (6th Cir. 1993).

The Board has held that in a Part 718 survivor's claim, the Administrative Law Judge must make a threshold determination as to the existence of pneumoconiosis under § 718.202(a) prior to considering whether the miner's death was due to the disease under § 718.205. *Trumbo v. Reading Antracite Co.*, 17 B.L.R. 1-85 (1993).

Medical Evidence

The Claimant was not represented in these claims by counsel or a representative, and she did not submit an Evidence Summary designating her submissions under the evidentiary restrictions of the regulations. The Director's Exhibits in the living miner's and survivor's claims were contained in a single, consecutively numbered file. At the hearing, counsel for the Director offered all 97 Exhibits in connection with both claims (Tr. 12). Although the Claimant did not specifically designate certain medical records that are contained in the Director's Exhibits, I have considered those exhibits that fall within the limitations on the Claimant's evidence.

Thus, with respect to the x-ray evidence, I have considered Dr. Baker's and Dr. Ahmed's interpretations as Mrs. H.'s affirmative x-ray evidence, and Dr. Poulos's interpretations of those two x-rays as the Employer's rebuttal evidence. Essentially, the x-ray evidence in the survivor's claim is identical to the x-ray evidence in the living miner's claim.

Pulmonary Function/Arterial Blood Gas Studies

As the Employer designated the pulmonary function and arterial blood gas studies from the living miner's claim (set out above), I have considered this evidence in connection with the survivor's claim (DX 57 – Living Miner's Claim).

Medical Reports

In connection with the survivor's claim, Mrs. H. submitted the treatment records from ARH, set out above, as well as Dr. Blake's autopsy report, also set out above. I consider Dr. Boll's letters to be treatment records, and thus I have considered them in connection with Mrs. H.'s survivor's claim. The Director also designated Dr. Blake's report as autopsy evidence. The Employer designated the reports by Dr. Dahhan as well as his deposition testimony, as set out above, as well as the following reports.

Dr. David M. Rosenberg

At the Employer's request, Dr. Rosenberg reviewed Mr. H.'s medical records and submitted a report dated August 21, 2006.[12] (EX 2, 3, 4 – Survivor's claim). Dr. Rosenberg noted that the pulmonary function test administered by Dr. Dahhan in 2003 did not show any restriction; Mr. H.'s oxygenation was normal, and his x-ray did not show micronodularity. Nor was there clinical evidence of pneumoconiosis in Mr. H.'s treatment records. While there was some anthracotic pigment (or anthracosis) noted on autopsy, this finding simply described black pigment deposition in the lungs, and not pathologic findings of pneumoconiosis. According to Dr. Rosenberg, the earliest pathologic finding of pneumoconiosis is the coal macule, which is associated with the formation of focal emphysema. He noted that anthracosis, or black pigment without coal macule formation, occurs in individuals living in an industrialized society, and does not represent pathologic pneumoconiosis.

Dr. Rosenberg noted that Mr. H. had a mild to moderate degree of obstruction when he was evaluated by Dr. Dahhan in 2003, which was not considered to be disabling. Toward the end of his life, Mr. H. developed congestive heart failure, with a left ventricular ejection fraction reduced to 20%. This condition disabled him from performing any type of arduous labor.

Dr. Rosenberg further discussed Mr. H.'s obstructive lung disease, noting that he had a significant degree of panlobular emphysema with bullous formation at autopsy. Dr. Rosenberg agreed that coal mine dust exposure can cause airflow obstruction, and specifically, emphysema. But when it does so, it begins in and around the coal macule as focal emphysema. As the coal macule evolves into micronodular, macronodular, and potentially progressive massive fibrosis, the associated emphysema can also worsen. A finding of bullous emphysema and panlobular emphysema, where extensive respiratory units of the lung are destroyed, represents the presence of emphysema related to past coal mine dust exposure.

But Dr. Rosenberg stated that Mr. H.'s chronic obstructive pulmonary disease did not represent the presence of legal pneumoconiosis. His COPD was not in and of itself disabling. Rather, he was disabled by his severe left ventricular heart failure, with an ejection fraction of

12 Dr. Rosenberg reviewed Dr. Dahhan's report, Dr. Boll's September 29, 2004 report, the Middlesboro-ARH records, the death certificate, Dr. Oesterling's report, and Dr. Blake's autopsy report. (DX 2, 3, 4).

20%. According to Dr. Rosenberg, this type of left ventricular failure was likely related to a hypertensive cardiomyopathy, and did not represent the presence of legal pneumoconiosis. In addition, the various areas of scarring with honeycombing did not represent either medical or legal pneumoconiosis, but was probably related to either past lung infections or chronic congestive heart failure. He noted that Dr. Oesterling found fungal organisms in Mr. H.'s lung tissue.

Dr. Rosenberg stated that Mr. H. obviously had severe endstage congestive heart failure, which was likely related to a hypertensive cardiomyopathy, but was not aggravated or hastened by exposure to coal mine dust, or pneumoconiosis. He concluded that while Mr. H. had moderate airflow obstruction, it was due to COPD caused by smoking, which also caused the development of bullous and panlobular emphysema.

Dr. Rosenberg testified by deposition on September 15, 2006 (EX 4). He noted that Mr. H. had a degree of obstruction on pulmonary function testing, and a significant degree of heart disease, with an ejection fraction of 20%, reflecting a marked compromise of left ventricular function, with enlargement of the left ventricle. These conditions caused various respiratory symptoms. But Mr. H. did not have evidence of either medical or legal coal workers' pneumoconiosis. According to Dr. Rosenberg, pathologically, Mr. H. had a very severe degree of emphysema, of the panlobular variety. He described this as a condition where the lung is destroyed and dilated, and replaced by nothing, like an empty space of nonfunctional lung. Mr. H.'s major problem was congestive heart failure; he also had various areas of scarring in his lungs, probably from old inflammatory changes or infection. He noted that Mr. H. had some fungal organisms in his lungs, as suggested by giant cell formation.

Although Dr. Rosenberg agreed that coal dust exposure can cause airflow obstruction, he noted that just because a particular miner has airway disease, it does not necessarily represent legal pneumoconiosis. He felt it was clear that Mr. H.'s obstructive lung disease was not related to coal mine dust. He noted the extensive degree of panlobular emphysema in his lungs pathologically, which represented areas of the lungs that were totally destroyed and emphysematous. Although coal mine dust exposure can cause emphysema, it is a different type, which begins in and around the coal macule as focal emphysema. But in Mr. H.'s case, there was advanced endstage emphysema without micronodular disease, or focal emphysema. There was only panlobular emphysema that related to cigarette smoking or other factors, but not exposure to coal mine dust.

Dr. Rosenberg stated that Mr. H.'s coal dust exposure would not have hastened his severe congestive heart failure, because there was no pathologic finding of a condition related to pneumoconiosis that would have aggravated his underlying heart disease.

Dr. Everett F. Oesterling, Jr.

At the Employer's request, Dr. Oesterling reviewed the histologic slides from the April 30, 2004 autopsy. (DX 79). Dr. Oesterling also testified by deposition on September 14, 2006 (EX 5). To illustrate and document his findings, Dr. Oesterling attached magnified photographs of the tissue slides. He pointed to the absence of visible black pigment in the multiple cross

sections of the lungs. He also prepared photomicrographs, which showed black pigment deposition, as well as collagen fibers, which comprise scarring. He indicated that this structure represented very early nodular formation, but did not attain a size to warrant a diagnosis of pneumoconiosis. Thus, although Mr. H. had anthracotic pigmentation in the subpleural tissues of his lungs, there was no evidence of interstitial coalworkers' pneumoconiosis.

Dr. Oesterling identified panlobular pulmonary emphysema on the slides, noting that the autopsy prosector also listed diffuse panlobular and bullous emphysema in his diagnosis. However, this type of emphysema is not typically associated with the inhalation of coal mine dust. Citing to the textbook "Pathology of the Lung," he stated that the emphysema that is seen in coal miners is specific to centriacinar emphysema, and unrelated to panacinar emphysema. Mr. H.'s emphysema could not be attributed to his exposure to coal mine dust, due to its type, and the very minimal anthracotic pigmentation.

Dr. Oesterling also described findings of left heart failure, resulting in extensive pulmonary congestion and intraalveolar hemorrhage. There was also an indication of some component of an infectious disease process. These processes resulted in very severe fibrotic change, which he characterized as end stage pulmonary fibrosis, also described as honeycombed lung. Dr. Oesterling pointed to emphysematous change and interstitial fibrosis in the left bronchi, with pigmentation typical in persons with significant inhalation of cigarette smoke.

Dr. Oesterling concluded that Mr. H. had significant interstitial pulmonary fibrosis, with areas of end stage honeycombing of the lungs. This was due to multiple factors, including hemorrhage into the lung, potentially infectious microorganisms, and cigarette smoke inhalation. But the limited anthracotic pigment appeared to be insufficient to have in any way contributed to this process; he noted that throughout the major areas of fibrotic change, the presence of silica crystals was not shown by partial polarized light.

In Dr. Oesterling's opinion, Mr. H. died of end stage congestive heart failure, resulting in cardiorespiratory arrest, acute renal failure, bilateral pleural effusions, and left lower lobe pneumonia, as stated on his death certificate. Although there was anthracosis in Mr. H.'s lungs, there was no evidence of the process of pneumoconiosis. Thus, coal workers' pneumoconiosis was in no way a factor in hastening, precipitating, or causing Mr. H.'s death.

Death Certificate

Finally, the record includes Mr. H.'s death certificate, which was completed by Dr. Maria Hortillosa (DX 76). She indicated that Mr. H.'s death was due to end stage congestive heart failure, cardio-respiratory arrest, acute renal failure, bilateral pleural effusion, pneumonia, and possible metastatic hepatic lesions.

DISCUSSION

Existence of Pneumoconiosis

I have evaluated the x-ray evidence in connection with the living miner's claim, and found that it did not establish that Mr. H. had pneumoconiosis. No additional x-ray evidence has been submitted in connection with the survivor's claim that would change my conclusion.

There are two autopsy reports in the record in connection with the survivor's claim: the report by Dr. Blake, the autopsy prosector, and the report by Dr. Oesterling, who reviewed Dr. Blake's report and examined the tissue slides. Both doctors found evidence of emphysema, but neither diagnosed coal workers' pneumoconiosis. Dr. Oesterling discussed the type of emphysema found in Mr. H.'s lungs, which is not caused by coal dust exposure, and attached medical literature to support this position.

Again, as in the living miner's claim, the existence of pneumoconiosis has not been established by use of the presumptions set out in §§ 718.304, 305, or 306.

The record in the survivor's claim includes the treatment records from ARH, and the reports and deposition testimony by Dr. Dahhan, discussed above. For the same reasons that I set out in the living miner's claim, I find that these records do not establish that Mr. H. had pneumoconiosis. For the same reasons as discussed above, I find that Dr. Boll's and Dr. Baker's opinions are not sufficient to establish that Mr. H. had pneumoconiosis.

Viewing all of the evidence relating to the existence of pneumoconiosis as a whole, I find that Mrs. H. has not established that Mr. H. had pneumoconiosis by a preponderance of the objective medical evidence. I also note that even if I were to find that Mrs. H. had established that Mr. H. had pneumoconiosis, she has not established that his death was caused by, contributed to by, or hastened by pneumoconiosis. The only physician to even suggest that this was so was Dr. Boll. However, there is no indication that Dr. Boll attended Mr. H. during his final illness, or that he had any first-hand knowledge of the circumstances surrounding his death. Dr. Boll did not review the other medical records, including the autopsy reports, and he did not offer any support for his conclusory statements. The physicians who treated Mr. H. during his final hospitalization did not implicate pneumoconiosis as a factor in his death, and it was not included on his death certificate. Finally, neither Dr. Fino, Dr. Dahhan, Dr. Blake, nor Dr. Oesterling identified pneumoconiosis as a factor in Mr. H.'s death.

Accordingly, I find that Mrs. H. is not entitled to benefits as a surviving spouse under the Act.

CONCLUSION

Because the evidence does not establish that Mr. H. suffered from pneumoconiosis, Mrs. H. is not entitled to benefits in the surviving spouse claim or the living miner's claim.

ORDER

It is hereby ORDERED that the survivor's claim for benefits under the Act is DENIED, and that the miner's claim for benefits under the Act is DENIED.

SO ORDERED.

A

LINDA S. CHAPMAN
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).